

Hepatic tumours induced by anabolic steroids in an athlete

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SUMMARY A fatal rupture of an hepatic tumour occurred in an athlete who had been taking anabolic steroids for several years as an aid to body building. The case illustrates the hazards of non-therapeutic androgen administration, and emphasises the need for athletes to be made clear of the disturbances to gonadal function, liver structure and function, and the threat to life.

Controlled administration of sex hormones may result in hepatic disease. The contraceptive pill is associated with peliosis hepatis, the number of symptomatic cases of focal nodular hyperplasia has increased considerably, and hepatic adenomas and carcinomas have also been reported.¹

Androgens may also produce peliosis hepatis, but usually they cause hyperplasia, multiple nodules, and tumours.¹ These changes were first recognised in patients with Fanconi's anaemia and other aplastic anaemias, but have also been seen in patients treated for hypopituitarism, impotence, and trans-sexualism.

We report an athlete who presented with a ruptured liver tumour; the histopathological findings were typical of those produced by the administration of androgen. The patient was taking anabolic steroids as an aid to body building.

Case report

A 27 year old Indian body builder, who had been taking anabolic steroids for at least three years was admitted to a district general hospital complaining of severe abdominal pain. There was no history of trauma. A laparotomy was performed, and extensive haemorrhage from the right lobe of the liver was seen. A right hepatic lobectomy was performed but the haemorrhage persisted and the patient died.

At post mortem examination, the musculature was noted to be extremely well developed. Internally, the major findings were in the abdominal cavity—the remaining left hepatic lobe was soft and friable and contained two tan coloured, circumscribed nodules, measuring 10 mm and 5 mm in diameter, respectively (fig 1). No bleeding point was identified. Elsewhere the

heart was hypertrophied, weighing 400 g, with a left ventricular wall thickness of 30 mm; and the testes were atrophic. The right hepatectomy specimen weighed 1070 g and contained two nodules, 25 mm and 40 mm in diameter. Extensive haemorrhage was associated with the large nodule.

Histological findings

The large nodule in the right hepatectomy specimen consisted of sheets of well differentiated cells resembling hepatocytes (fig 2). There was an area of central haemorrhage surrounded by groups of cells forming acini containing bile pigment, the cells having occasional bizarre nuclei and rare mitoses (fig 3). The nodule was well circumscribed with no evidence of vascular invasion.

The smaller nodule was also composed of sheets of hepatocyte-like cells, but there was no acinar formation or pleomorphism. The two nodules identified at necropsy had a similar appearance, and hepatocellular adenoma was diagnosed. The acinar formation,

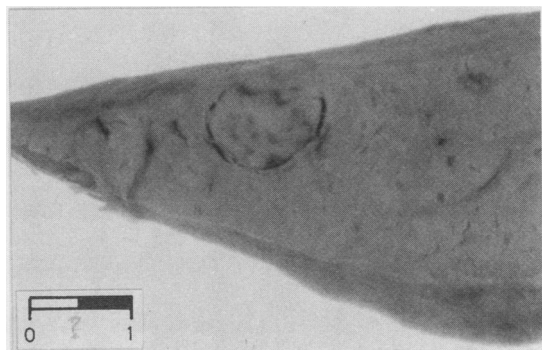


Fig 1 Hepatic nodule.

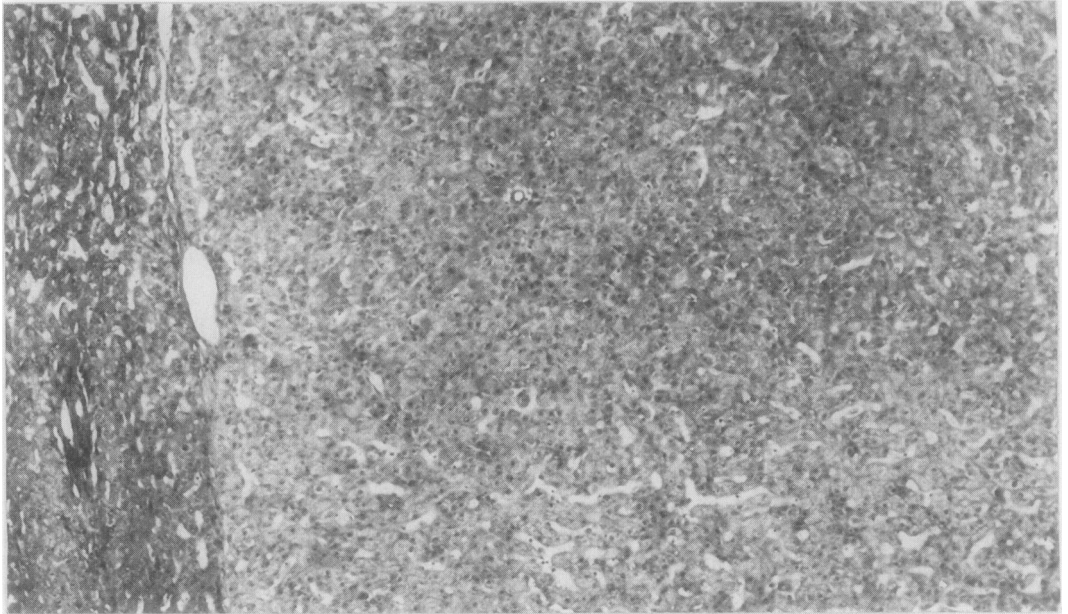


Fig 2 Edge of hepatic adenoma and adjacent hepatic parenchyma. (Haematoxylin and eosin.)

pleomorphism, and occasional mitoses seen in the larger nodule, however, were regarded as being suggestive of malignant transformation, although immunocytochemical staining for α fetoprotein was negative. The remaining liver showed normal vascular

relations but lobular hyperplasia and ectatic sinusoids were noted.

Discussion

In man, continuous treatment with anabolic steroids

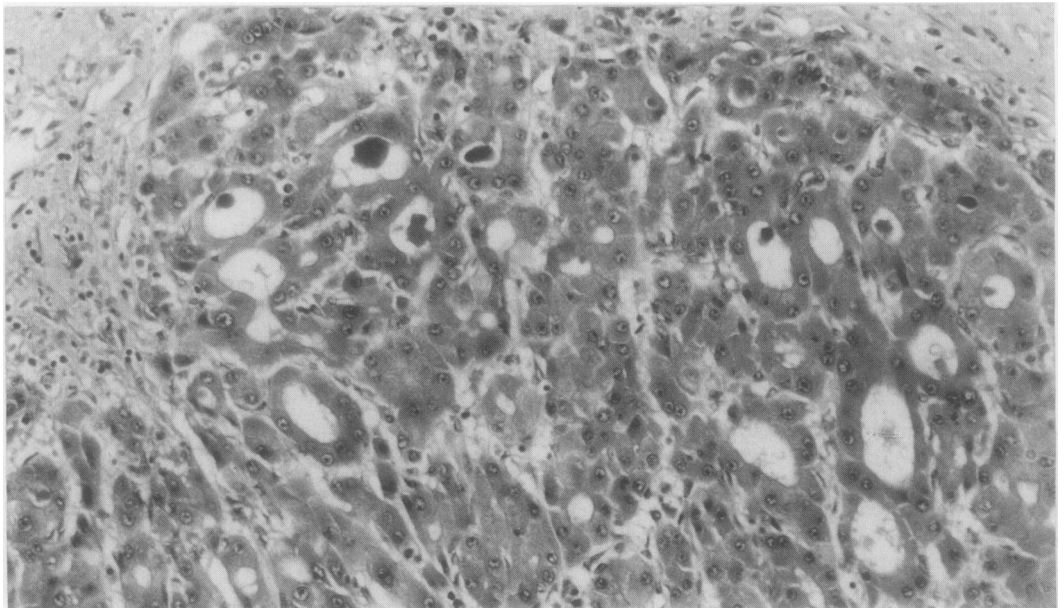


Fig 3 Acinar formation with central bile plugging and nuclear pleomorphism. (Haematoxylin and eosin.)

regularly produces hyperplasia,² and hepatic nodules are often found in patients dying of intercurrent illness. Whether these nodules can be regarded as true tumours is contentious as they may regress when the steroids are discontinued.^{3,4} Convincing evidence of metastasis has not been produced, and although some authors have regarded them as hepatomas, this has been disputed.⁵

The use of anabolic steroids is thought to be widespread among athletes: the main hazard seems to be rupture of the liver nodules, which has previously been described⁶ and which was the cause of death in this case. The long term risk of malignancy is hard to assess. Evidence of interference with testicular function was also present in this case, presumably as a result of pituitary gonadotrophin suppression.

Athletes and body builders should be made aware of the predictable disturbance of gonadal function, of the regular disturbance of liver structure and function, and of the potential hazard to life.

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